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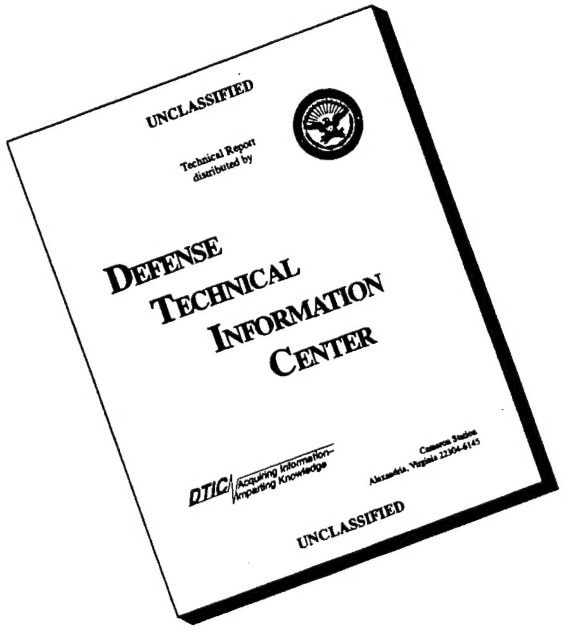

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ALVEOLAR BONE AS A FUNCTION OF AGE AND PERIODONTAL DISEASE: HISTOMORPHOMETRIC ANALYSIS IN THE BABOON

A THESIS

Presented to the Faculty of

The University of Texas Graduate School of Biomedical Sciences

at San Antonio

in Partial Fulfillment of the Requirements for the Degree of

MASTER OF SCIENCE

By

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San Antonio, Texas

May 1995

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Age-related changes in soft tissues of the periodontium have been investigated, yet less attention has been directed to changes occurring in the alveolar bone. Presumably, some of these changes would include quantifiable alterations in alveolar bone density and cellularity. Periodontal disease and osteoporosis are two pathologies whose increasing prevalence among aged individuals is well-documented. A relationship between these disease entities has been conjectured; however, no research firmly establishes an association. Because the baboon demonstrates both naturally-occurring osteoporosis and periodontal

disease, this model is highly appropriate for the study of age-related changes in alveolar bone. The purpose of the present investigation was to characterize baboon alveolar bone by Quantitative Histomorphometric Analysis (QHA) with reference to age and histologic parameters for periodontal disease. Mandibles were collected at necropsy from 21 skeletally mature female baboons (*Papio cynocephalus anubis*) 8 to 30 years of age (human age equivalent: 24 to > 100 years). Bilateral block sections of first and second molars were sagittally sectioned. 2 mm thick sections representing interproximal bone in the contact area were decalcified, paraffin-embedded, step-serial sectioned in a mesio-distal plane at 80 μ intervals, 6 μ thick. Histomorphometric analysis was performed by one examiner using a calibrated ocular grid mounted in a light microscope with a point counter linked to a microcomputer. QHA findings confirmed an increase in naturally occurring attachment loss with age ($r = 0.541$, $p = 0.011$), decreasing osteocyte density in apical bone with age ($r = -0.634$, $p = 0.0027$) and decreasing active osteoblastic surfaces in crestal bone both with age ($r = -0.655$, $p = 0.0017$) and with attachment loss ($r = -0.47$, $p = 0.035$). Despite an age-related decrease in bone cell density and blastic activity, there was no evidence of a decrease in total bone volume in crestal or apical alveolar bone with either increasing age or attachment loss. This non-human primate model demonstrated that alveolar bone appears to resist osteoporotic changes despite advancing age or periodontal disease, suggesting that teeth may impart forces to bone which mask normal density changes evident with aging. Different parameters may be necessary for analysis of alveolar bone than those used for systemic bone disease.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

I. INTRODUCTION AND LITERATURE REVIEW

Age-related changes in alveolar bone have been investigated, yet well-documented histomorphometric studies in alveolar bone are lacking (1). Reported observations of human alveolar bone changes with age (2) include diminished vascularity due to diffuse calcification of nutrient canals (3), increasing number of interstitial lamellae with fewer cells in the osteogenic layer (4), decreased trabeculation and osteoporosis (5). Difficulty lies in distinguishing between those changes induced by disease and those resulting from the normal, physiologic effects of senescence (6). Beyond qualitative assessment, definitive data regarding age-associated changes in alveolar bone density, cell populations and activity are limited or unavailable.

Quantitative Histomorphometric Analysis (QHA) is a precise and valuable tool which utilizes biopsy of bone tissue for evaluation of disturbances in osseous remodeling. QHA is the only method that provides direct and precise analysis of the cellular and tissue status of cortical bone and the trabecular structural unit in the medullary compartment (7). Although an invasive technique, QHA is the accepted "gold standard" for assessing bone matrix density and remodeling and provides quantitative assessment of bone status over that currently possible through clinical exam or noninvasive imaging techniques including Digital Subtraction Radiography, Computerized Tomography or Dual-photon Absorptiometry.

Models for study of periodontal disease have utilized non-human primates, taking advantage of their close anatomic and biologic similarities with humans (8, 9). Available data suggest the baboon comes closest to matching the human dentition in terms of dental formula, tooth size, morphology, occlusion and histology

(8). Spontaneous gingivitis and periodontitis increases in severity with baboon age (10, 11) and is characterized by the same inflammatory infiltrate observed in that of humans (8). Because the baboon furthermore demonstrates both naturally-occurring skeletal and maxillofacial bone loss (12), this non-human primate may be ideally suited to study age-related change in alveolar bone.

The purpose of this study was to investigate age-associated changes in baboon alveolar bone by methodology used in the study of systemic bone disease, QHA, relative to established histologic parameters for periodontal disease.

II. MATERIALS AND METHODS

A. Study Population

Materials for this study were provided by the Southwest Foundation for Biomedical Research in San Antonio, Texas. Mandibles were collected at necropsy from 21 skeletally-mature female baboons, *Papio cynocephalus anubis,* 8 to 30 years of age with a human age equivalent (HAE) of 24 to > 100 years. These animals had been euthanized for humane reasons due to injury or because they could no longer protect themselves within the colony. Necropsy findings revealed no evidence of disease; however, older females exhibited prominent phenotypic changes suggestive of osteoporosis including loss of height, lumbar kyphosis and spondylosis.

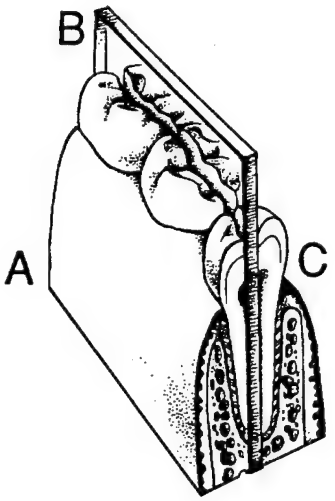
B. Tissue Processing

Mandibles were fixed in 10% neutral-buffered formalin, processed in standard fashion and stained with hematoxylin-eosin (HE) for histomorphometric analysis (12). Bilateral block sections of first and second molars were sagittally sectioned (Figure 1) with an alcohol-cooled Buehler Isomet saw and a diamond wafering blade. Two-mm thick sections representing interproximal bone in the contact area were decalcified with ethylenediamine tetra acetic acid (EDTA), embedded in paraffin (Figure 2), step-serial sectioned 6 μ thick in a mesio-distal plane at 80 μ intervals.

C. Quantitative Histomorphometric Analysis (QHA)

QHA was performed by one examiner (KN) using a calibrated ocular grid (Zeiss Intergrationsplatte II 100/25) mounted in a light microscope with a point-counter, digitizing tablet and light-emitting diode cursor linked to a microcomputer, using the Bioquant™ IV Image Analysis System (R&M Biometrics, Inc.). Three representative sections from each interproximal site were analyzed. Using the

Figure 1. Plane of Mandibular Section. Bilateral block sections of first and second molars were sagittally sectioned to yield 2-mm thick sections containing interproximal bone in the contact area. Graphic reprinted with permission of Dr. James Vacek and Quintessence Publishing Company, Inc. (13).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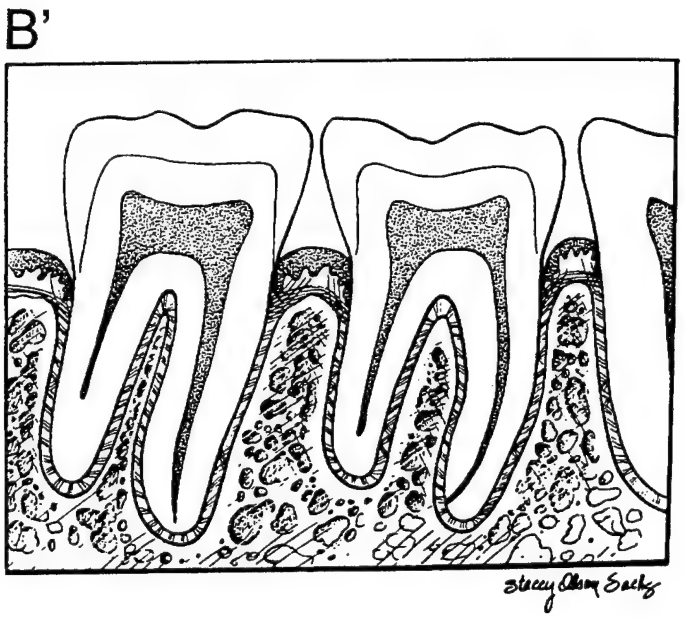

B'

Figure 2. Tissue Specimen in Cassette. Two-mm thick sections placed in cassette for paraffin-embedding and mesio-distal step-serial sectioning at 80 μ intervals.

cemento-enamel junction (CEJ) as a reference, linear measurements were made at x22 magnification at the mesial and distal aspects of the interproximal bone to determine the amount of alveolar crest resorption (ACL), periodontal attachment loss (Attch Loss) and the connective tissue attachment (CT Attch) dimension (Figure 3). Interproximal distance was measured at 3 sites to test for any confounding influence of root proximity on morphometric parameters (Figure 4). The entire area of interproximal bone was traced and measured in mm^2. Along a line bisecting the interproximal bone, QHA was completed on 3 non-overlapping fields of bone at x215 magnification in the crestal as well as the subjacent apical 2 mm (Figure 5). At each interproximal site, 18 fields of bone (3 sections x 6 fields per section), each having an area of 0.136 mm^2 (0.37 mm x 0.37 mm) were analyzed. Alveolar bone density was determined by estimating total bone volume (TBV, Figure 6) by a point counting technique (14). Other parameters included osteoclast and osteocyte density, osteoblastic and osteoclastic surfaces (Figures 7-9).

D. Data Analysis

Data were analyzed with Statistical Analysis Systems software (SAS, version 6.6) using both the site and the animal as the unit of measurement. Correlation between age and histomorphometric variables was tested by Pearson Correlation Coefficient/Probability and Analysis of Variance (ANOVA) of Regression. Type III ANOVA tested for bony changes attributable to aging, attachment loss, or other morphometric parameters. The student *t*-test was used to test for differences by degree of attachment loss and age category. Intra-examiner error was tested prior to the analysis by performing three sets of measurements, 72 hours apart, on four randomly chosen specimens. Cronbach's Coefficient Alpha was utilized to determine intra-examiner error for each parameter.

Figure 3. Determination of Crestal Resorption, Attachment Loss and Connective Tissue Attachment. Alveolar crest level (ACL): CEJ to the alveolar crest (AC). Attachment loss (Attch Loss): CEJ to apical cell of the junctional epithelium (JE). Connective tissue attachment (CT Attch): apical cell of JE to AC. (HE x 22).

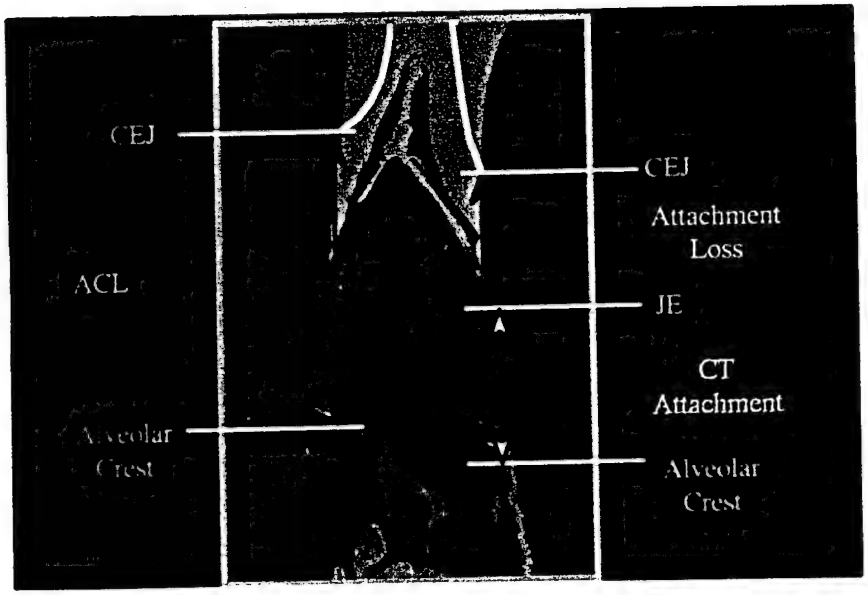

CEJ
ACL
Alveolar Crest
CEJ
Attachment Loss
JE
CT Attachment
Alveolar Crest

Figure 4. Measures of Interproximal Distance. Interproximal distance was measured at the most apical CEJ (C2), at the alveolar crest (C7) and at a standard distance, 3.7 mm apical to the CEJ (C4). (HE x 22).

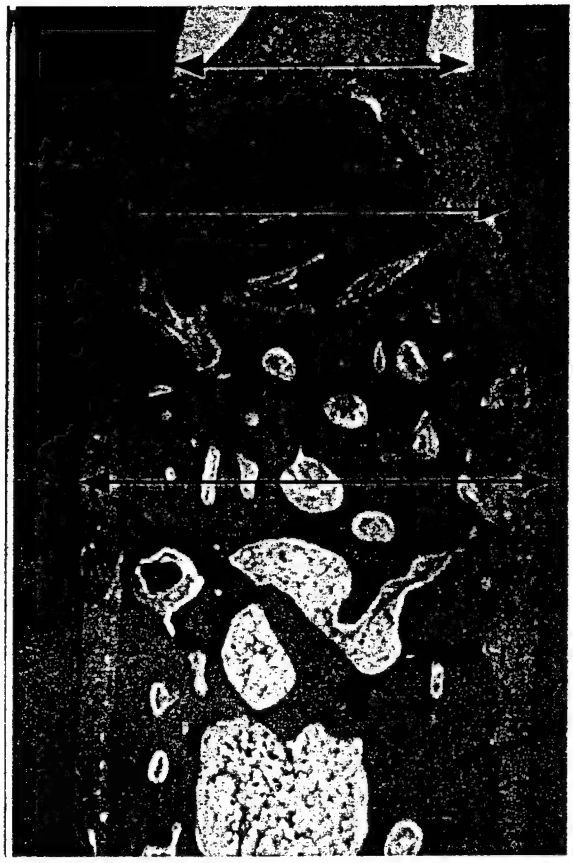


Figure 5. Crestal and Apical QHA Fields. Along a superimposed bisecting line, QHA was performed on 3 fields of bone in the crestal and subjacent apical 2 mm regions. Each field has an area of 0.136 mm^2 (0.37 mm x 0.37 mm). (HE x 22).

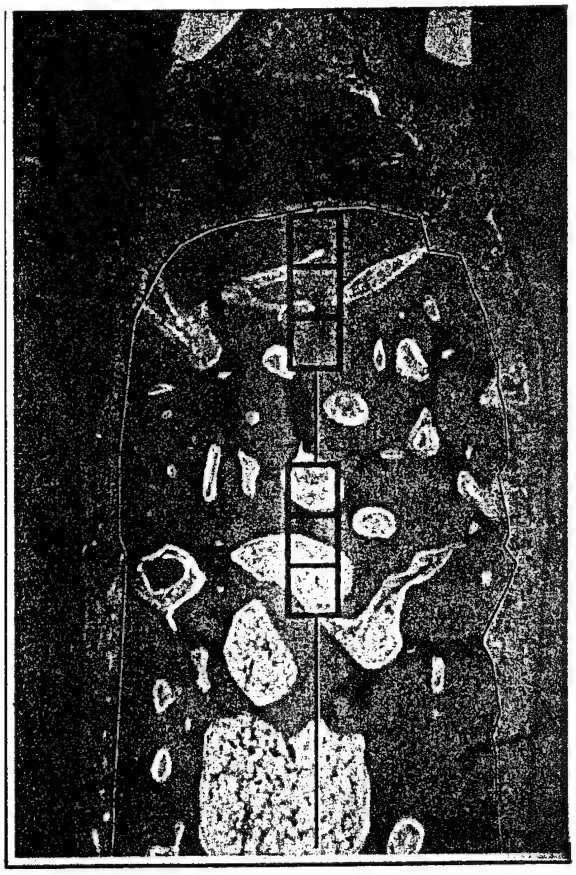
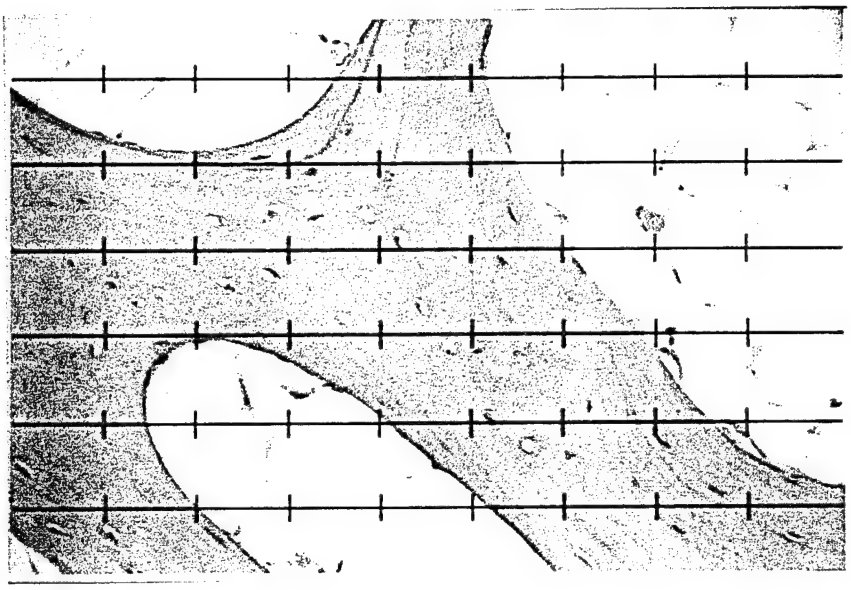


Figure 6. Total Bone Volume (TBV). A portion of the grid used for morphometric analysis is shown. Grid-lines intersect with points, or hits superimposed over bone. The grid comprises 100 possible hits with each square representing an area equal to 1 percent. The number of hits over bone affords a percentage measure of alveolar bone density. TBV represents the total volume of bone matrix including cortical and cancellous bone (7). (HE x 215).

Figure 7. Osteocyte Density. The total number of osteocytes in lacunae per field were counted to determine osteocyte density relative to TBV. (HE x 215)



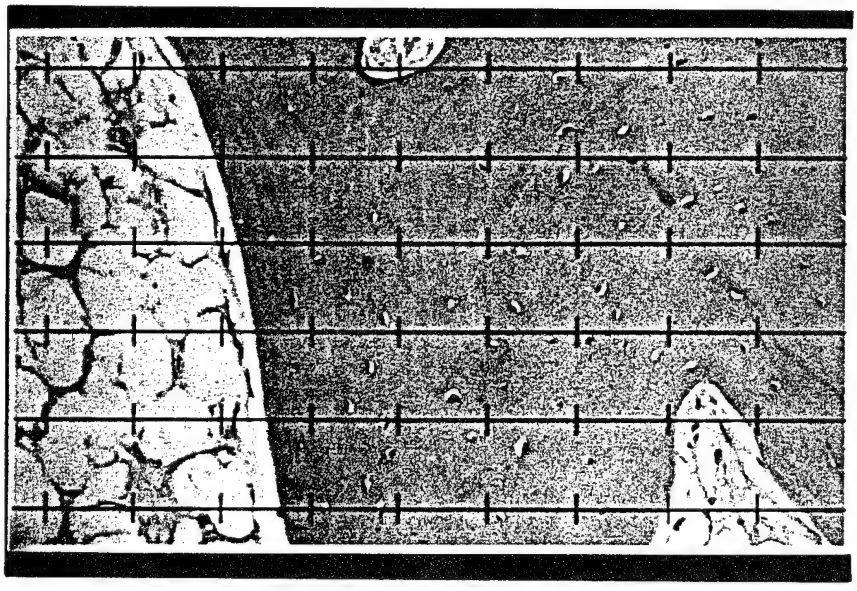
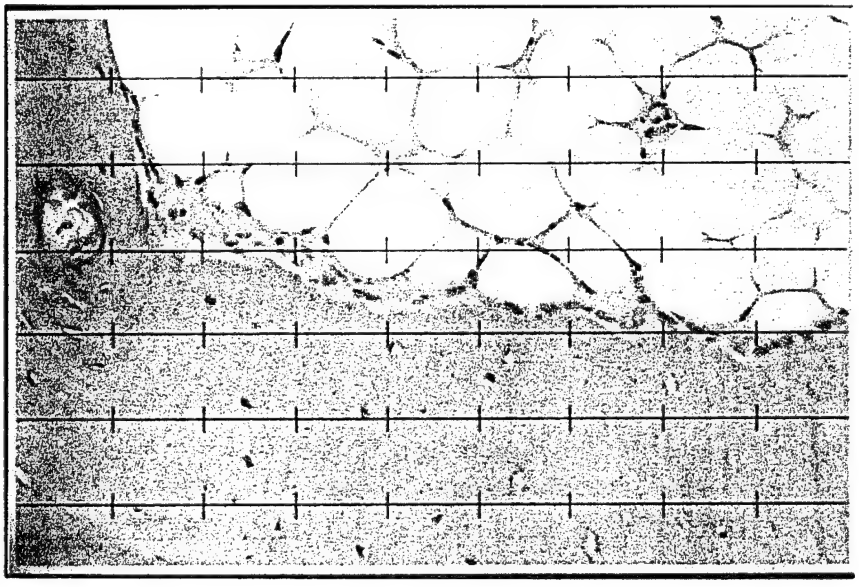


Figure 8. Osteoblastic Surfaces. The number of horizontal lines intersecting with active osteoblastic surfaces was counted, as a percentage of total intersects with bone. (HE x 215).

Figure 9. Osteoclastic Surfaces. Horizontal lines intersecting with resorbing surfaces were assessed, as a percentage of total intersects with bone. (HE x 215)



III. RESULTS

A. Histologic Parameters

Mean histologic measurements for the study group are summarized in Table 1. Linear measurements (Attch Loss, ACL and CT Attch) represent the average of measurements made at the mesial and distal of each interproximal bone site. Due to the high correlation between right and left sides of the mandible ($r = 0.83$) and non-independence of sites within the same arch (15), side measurements were averaged. Mean Attch Loss was 0.9 mm and ranged from 0.06-2.36 mm. ACL, a measure of crestal resorption, ranged from 1.12-4.06 mm with a mean of 2.44 mm. CT Attch averaged 1.61 mm in length (range 0.74-2.92 mm).

TBV and cellular parameters, reported separately for crestal and subjacent apical regions of interproximal bone, are the average of right and left sides of the mandible (Table 1). Mean osteocyte density was 85.4 cells/TBV in the crestal region versus 91.6 cells/TBV in the apical region. A number of significant correlations were noted between particular histologic parameters and aging (Table 2), attachment loss (Table 3) and interproximal distance (Table 4).

1. Attachment Loss. Histologic findings confirmed increasing attachment loss with animal age, significant using either the site ($r = 0.547$, $p = 0.0004$, Figure 10) or the animal ($r = 0.54$, $p = 0.01$, Figure 11) as the unit of measurement. When animals were segregated by age into a young group, 8-13 years (HAE $\leq$ 39 years) and an old group, 15-30 years (HAE $\geq$ 54), mean attachment loss significantly increased in the older cohort (Table 5 and Figure 12). Attachment loss was also significantly correlated with interproximal distance (C4) measured at a standard distance (3.7 mm) from the most apical CEJ ($r = -0.52$, $p = 0.018$, Table 4). Thus, as the distance between adjacent teeth decreased, attachment loss increased.

Table 1. HISTOLOGIC PARAMETERS

Linear Measurements (n=20)	**Mean**	**Std Err**	**Minimum**	**Maximum**
Attachment Loss (mm) n=21	0.903	0.138	0.062	2.365
Alveolar Crest Level (mm)	2.443	0.219	1.117	4.057
CT Attachment (mm)	1.607	0.141	0.735	2.916
Interproximal Dx C4 (mm)	1.521	0.113	0.672	2.452
Crestal Interproximal Dx C7 (mm)	1.449	0.083	0.617	2.109
Animal Age (years) n=21	18.190	1.412	8.000	30.000
CRESTAL (n=20)	**Mean**	**Std Err**	**Minimum**	**Maximum**
Crestal TBV (Hits over bone)	74.411	2.565	45.667	89.833
Crestal Osteocytes/TBV	85.375	3.786	55.420	113.961
Crestal Osteoclasts/TBV	0.268	0.167	0.000	3.365
% Crestal Blastic Surfaces	3.957	1.089	0.000	18.631
% Crestal Resorptive Surfaces	1.040	0.420	0.000	6.790
APICAL (n=20)	**Mean**	**Std Err**	**Minimum**	**Maximum**
Apical TBV (Hits over bone)	61.239	4.397	24.889	91.000
Apical Osteocytes/TBV	91.590	4.952	62.208	130.778
Apical Osteoclasts/TBV	0.032	0.019	0.000	0.338
% Apical Blastic Surfaces	2.765	1.074	0.000	15.732
% Apical Resorptive Surfaces	0.144	0.078	0.000	1.398

Table 2. CORRELATION WITH AGING

	r	p
Attachment Loss, n=21	0.54	0.011 * †
Alveolar Crest Level, n=36	0.39	0.018 (ϕ) †
Apical Osteocytes/TBV, n=20	- 0.63	0.002 * †
% Crestal Blastic Surfaces, n=20	- 0.65	0.001 * †
Ct Attachment, n=20	- 0.014	0.95 (ϕ)(NS)
Crestal TBV, n=36	- 0.159	0.35 (ϕ)(NS)
Apical TBV, n=36	- 0.122	0.47 (ϕ)(NS)

* $p < 0.05$; significant at animal level

(ϕ) not significant at animal level; $p > 0.05$

† $p < 0.05$; significant at site level

(NS) not significant at site level; $p > 0.05$

Table 3. CORRELATION WITH ATTACHMENT LOSS

	r	p
Age, n=21	0.54	0.011 * †
CT Attachment, n=20	0.33	0.146 (ϕ)(NS)
% Crestal Blastic Surfaces, n=20	- 0.47	0.035 * †
Alveolar Crest Level, n=20	0.79	0.0001* †
Interproximal Dx (C4), n=20	- 0.52	0.018 * †
Crestal TBV, n=36	- 0.23	0.17 (ϕ)(NS)
Apical TBV, n=36	0.128	0.45 (ϕ)(NS)

* $p < 0.05$; significant at animal level

(ϕ) not significant at animal level; $p > 0.05$

† $p < 0.05$; significant at site level

(NS) not significant at site level; $p > 0.05$

C4 = 3.7 mm apical to CEJ

Table 4. CORRELATION WITH INTERPROXIMAL DX (C4)

	r	p	
Crestal TBV, n=36	- 0.135	0.40	(ϕ)(NS)
Apical TBV, n=36	- 0.278	0.10	(ϕ)(NS)
% Crestal Blastic Surfaces, n=36	0.36	0.028	(ϕ) †
Attachment Loss, n=21	- 0.52	0.018	* †
Alveolar Crest Level; n=20	-0.51	0.02	* †

* $p < 0.05$; significant at animal level

(ϕ) not significant at animal level; $p > 0.05$

† $p < 0.05$; significant at site level

(NS) not significant at site level; $p > 0.05$

C4 = 3.7 mm apical to CEJ

Figure 10. Change in Mean Attachment Loss With Age (n=37).
Using the site as the unit of measurement, mean attachment loss increased with age.

CHANGE IN MEAN ATTACHMENT LOSS

r=0.547, p=0.0004, n=37, y=0.054x-0.139

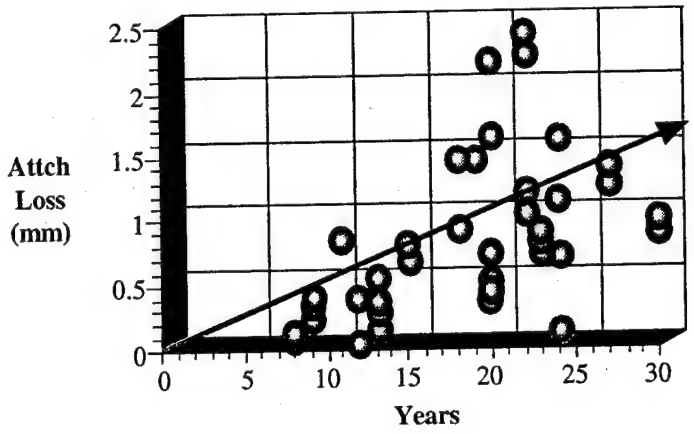


Figure 11. Change in Mean Attachment Loss With Age (n=21). Using the animal as the unit of measurement, mean attachment loss increased with age.

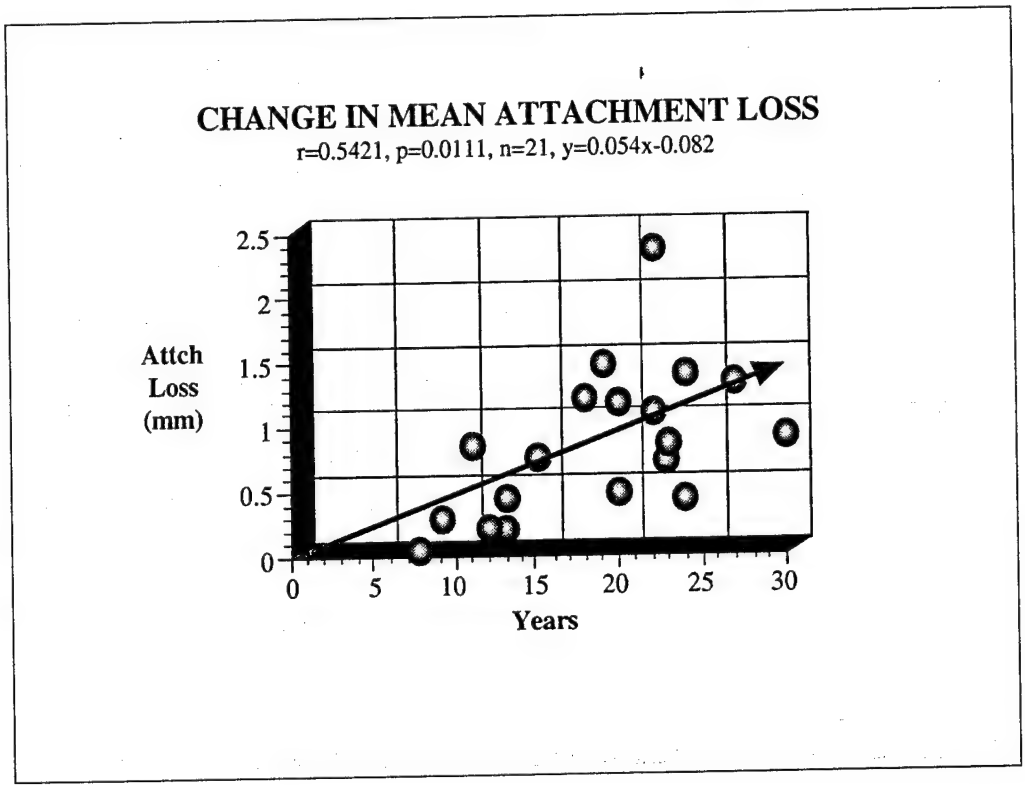

CHANGE IN MEAN ATTACHMENT LOSS
r=0.5421, p=0.0111, n=21, y=0.054x-0.082
Attch Loss (mm)
2.5
2
1.5
1
0.5
0
0
5
10
15
20
25
30
Years

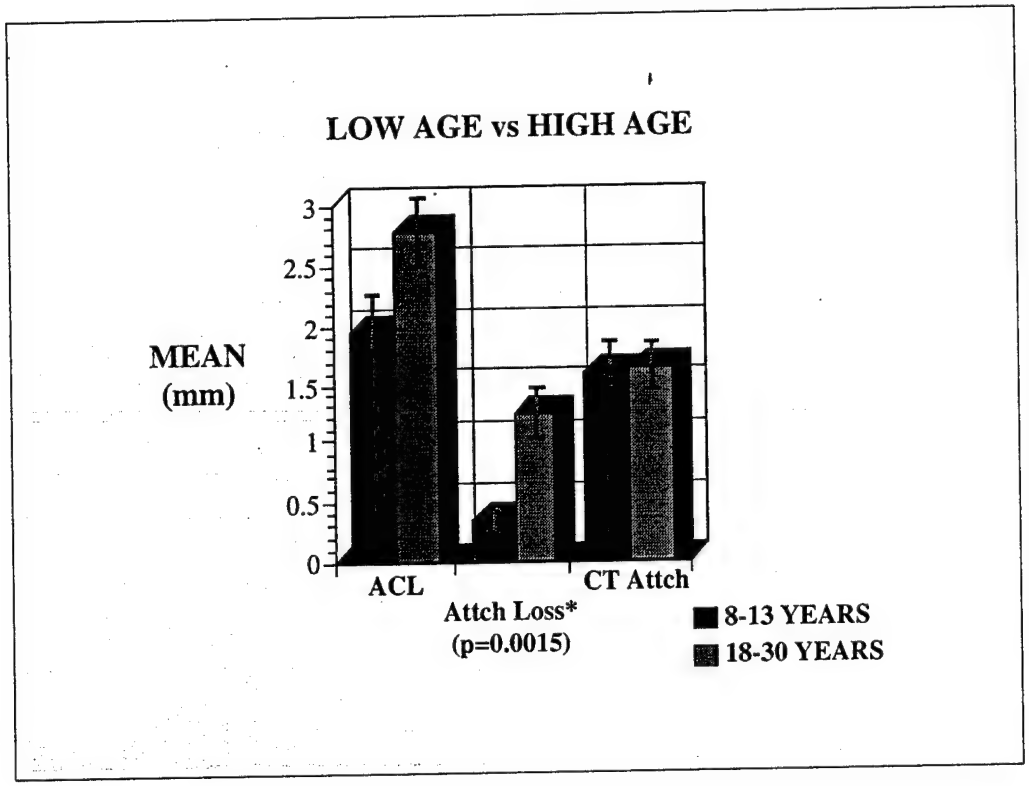
Table 5. LOW AGE VERSUS HIGH AGE

	8-13 Years, n=7 HAE 24-39 Years		18-30 Years, n=12 HAE 54 to > 100 Years		
Variable	Mean	Std Err	Mean	Std Err	p
Alveolar Crest Level, mm	1.95	0.33	2.77	0.28	0.086
Attachment Loss, mm	0.34	0.10	^1.21	0.16	0.002 *
CT Attachment, mm	1.61	0.24	1.64	0.19	0.930
Crestal TBV	77.42	2.96	72.14	3.86	0.357
Apical TBV	67.06	6.32	56.99	6.22	0.304
Crestal Cytes/TBV	87.61	6.09	81.69	4.67	0.452
Apical Cytes/TBV	105.90	6.05	79.98	4.92	0.005 *
% Crestal Blastic Surfaces	7.05	2.24	1.45	0.44	0.047 *
% Apical Blastic Surfaces	3.92	2.14	1.90	1.30	0.400
% Crestal Clastic Surfaces	0.55	0.30	1.41	0.67	0.255
% Apical Clastic Surfaces	0.24	0.20	0.52	0.07	0.522
Crestal Clasts/TBV	0.12	0.07	0.38	0.27	0.369
Apical Clasts/TBV	0.06	0.05	0.02	0.01	0.441

*$p < 0.05$

^ n=13

Figure 12. Low Versus High Age. Attachment loss was significantly increased in old animals (18-30 years of age; HAE $\geq$ 54 years) compared to young animals (8-13 years of age; HAE $\leq$ 39 years). Mean alveolar crest level and CT attachment were not significantly different between age groups.


LOW AGE vs HIGH AGE
MEAN
(mm)
3
2.5
2
1.5
1
0.5
0
ACL
CT Attch
Attch Loss*
(p=0.0015)
8-13 YEARS
18-30 YEARS

2. Alveolar Crest Level. ACL indicated that crestal resorption increased with animal age at the site level ($r = 0.39$, $p = 0.018$, Table 2) but not at the animal level ($r = 0.32$, $p = 0.15$). Furthermore, no significant differences were noted in crestal resorption between young and old animal groups (Table 5). Mean crestal resorption increased with increasing attachment loss ($r = 0.79$, $p = 0.0001$) at both the site and animal level (Table 3). When animals were grouped according to severity of attachment loss (< 0.5 mm versus > 1 mm) there was a significant difference in ACL (Figure 13 and Table 6). ACL was also significantly correlated with interproximal distance (C4) measured at a standard distance (3.7 mm) from the most apical CEJ ($r = -0.51$, $p = 0.02$, Table 4).

3. Total Bone Volume. QHA revealed a slight trend for alveolar density (TBV) to decrease with age (Figure 14), yet this tendency was not significant either in crestal ($r = -0.159$, $p = 0.35$) or apical bone ($r = -0.122$, $p = 0.47$, Table 2). Mean TBV did not significantly differ between young and old animal cohorts (Figure 15, Table 5). Furthermore, TBV poorly correlated with increasing attachment loss ($r = -0.014$, $p = 0.95$, Table 3). When animals were grouped according severity of attachment loss (< 0.5 mm versus > 1 mm) there was no significant difference in mean TBV between the 2 categories of attachment loss (Figure 16, Table 6). TBV was not significantly correlated with interproximal distance (Table 4).

4. Bone Cellularity. The concentration of Apical Osteocytes significantly decreased with age ($r = -0.634$, $p = 0.0027$, Figure 17, Table 2). Mean osteocytes decreased in the older age group in apical bone ($p = 0.0045$, Figure 18, Table 5). This change was exemplified by highly cellular apical bone in an 8 year-old baboon (Figure 19) versus sparse osteocytes observed in a 27 year-old animal (Figure 20). There was no significant difference between Osteoclasts/TBV in crestal or apical

Figure 13. Low Versus High Attachment Loss. ACL was significantly increased in animals with greater attachment loss. CT attachment was not significantly different when animals were grouped by severity of attachment loss.

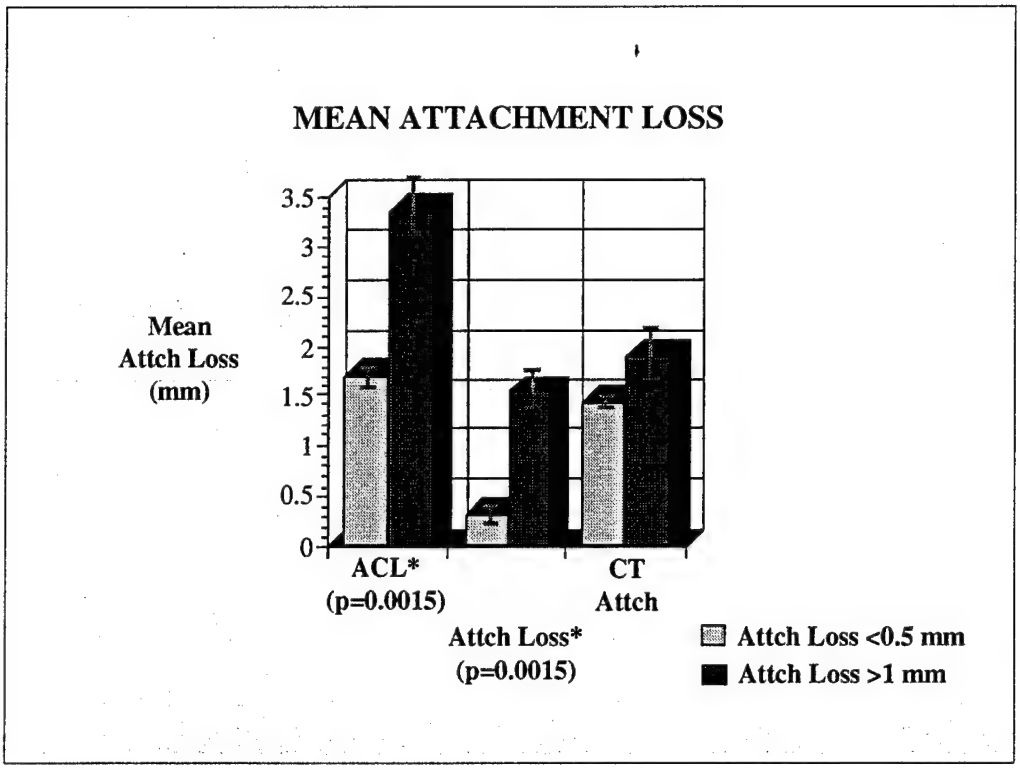

MEAN ATTACHMENT LOSS
Mean Attch Loss (mm)
3.5
3
2.5
2
1.5
1
0.5
0
ACL* (p=0.0015)
CT Attch
Attch Loss* (p=0.0015)
Attch Loss <0.5 mm
Attch Loss >1 mm

Table 6. LOW VERSUS HIGH ATTACHMENT LOSS

	Low Attch Loss, n=8 (< 0.5 mm)		High Attch Loss, n=7 (>1 mm)		
Variable	Mean	Std Err	Mean	Std Err	p
Age, years	13.50	2.01	^21.5	1.04	0.003 *
Alveolar Crest Level, mm	1.72	0.13	3.36	0.32	0.0015 *
Attachment Loss, mm	0.31	0.05	^1.54	0.17	0.0001 *
CT Attachment, mm	1.41	0.11	1.92	0.28	0.131
Crestal TBV	78.60	2.39	69.76	6.41	0.234
Apical TBV	61.39	7.46	64.70	7.00	0.754
Crestal Cytes/TBV	84.90	6.52	82.04	4.95	0.739
Apical Cytes/TBV	98.95	7.87	80.87	8.14	0.135
% Crestal Blastic Surfaces	5.74	2.12	1.48	0.62	0.090
% Apical Blastic Surfaces	1.52	0.79	2.92	2.19	0.566
% Crestal Clastic Surfaces	0.40	0.25	1.63	0.95	0.252
% Apical Clastic Surfaces	0.21	0.17	0.17	0.11	0.858
Crestal Clasts/TBV	0.09	0.06	0.57	0.47	0.343
Apical Clasts/TBV	0.05	0.04	0.03	0.02	0.703

*$p < 0.05$

^ n=8

Figure 14. Change in Total Bone Volume With Age. QHA revealed a slight trend for TBV to decrease some 10% with age, but this tendency was not significant either in crestal bone ($r = -0.159$, $p = 0.35$) or apical bone ($r = -0.122$, $p = 0.47$).

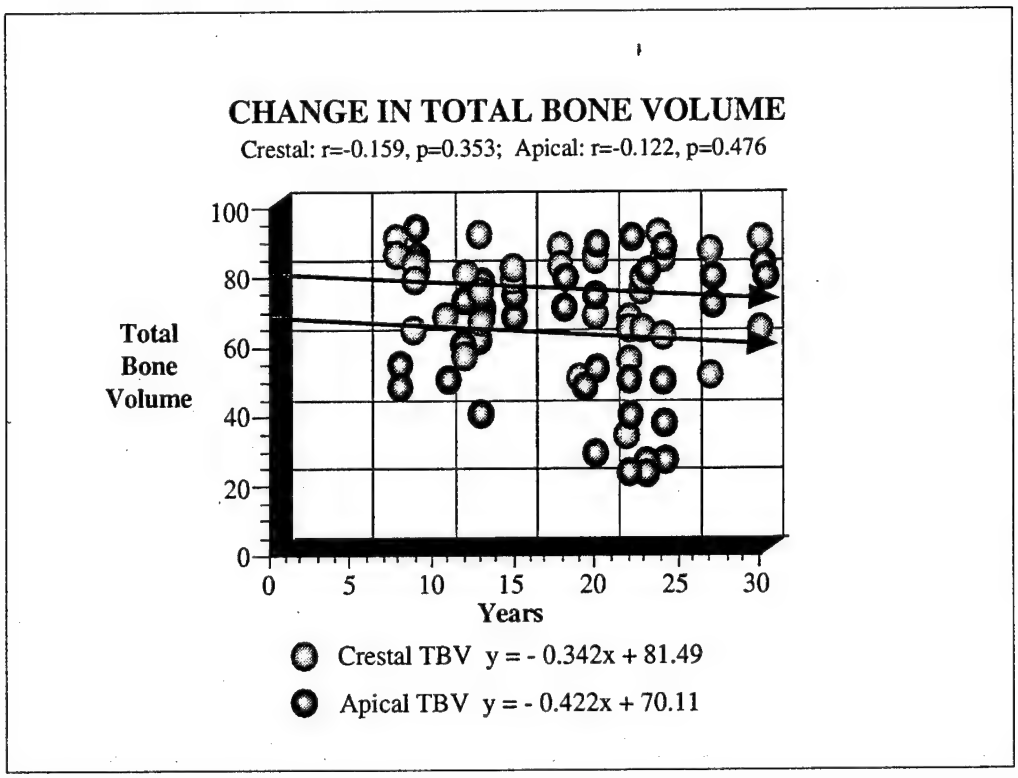

CHANGE IN TOTAL BONE VOLUME
Crestal: r=-0.159, p=0.353; Apical: r=-0.122, p=0.476
Total Bone Volume
100
80
60
40
20
0
0
5
10
15
20
25
30
Years
Crestal TBV y = - 0.342x + 81.49
Apical TBV y = - 0.422x + 70.11

Figure 15. Total Bone Volume By Age Group. Mean TBV was not significantly different in old animals (18-30 years of age; HAE $\geq$ 54 years) compared to young animals (8-13 years of age; HAE $\leq$ 39 years) in either crestal or apical bone.

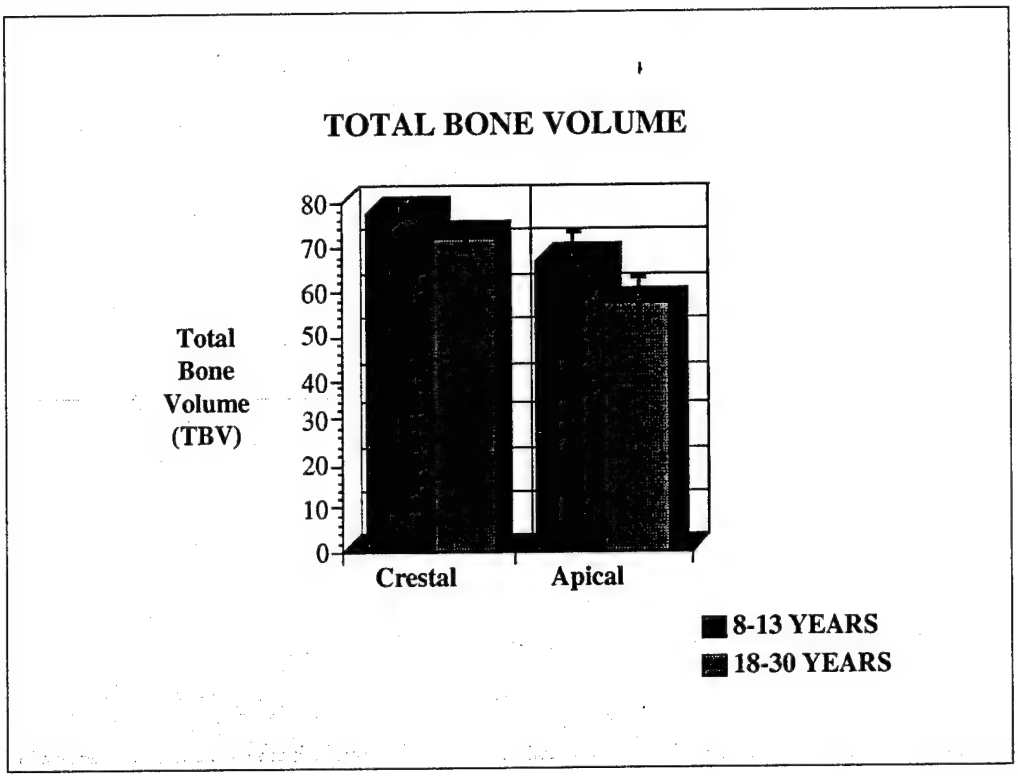

TOTAL BONE VOLUME
Total Bone Volume (TBV)
80
70
60
50
40
30
20
10
0
Crestal
Apical
8-13 YEARS
18-30 YEARS

Figure 16. Total Bone Volume By Attachment Loss Group. There was no significant difference in mean TBV between the 2 categories of attachment loss (< 0.5 mm versus > 1 mm) in crestal or apical bone.

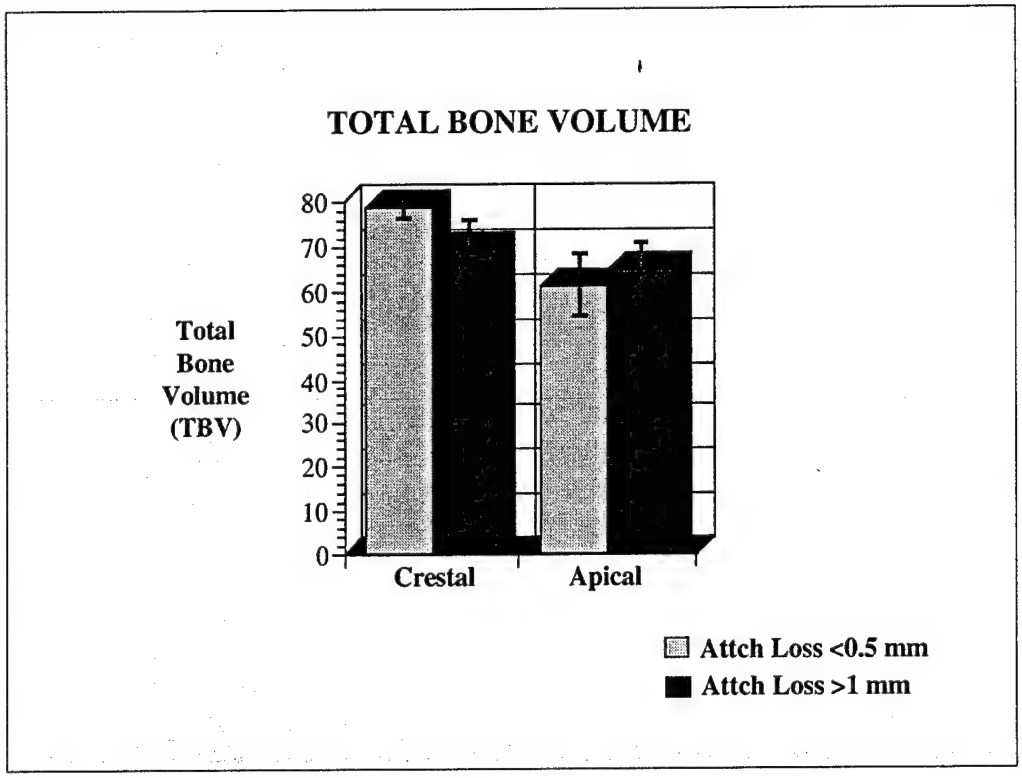

TOTAL BONE VOLUME
Total Bone Volume (TBV)
80
70
60
50
40
30
20
10
0
Crestal
Apical
Attch Loss <0.5 mm
Attch Loss >1 mm

Figure 17. Change in Mean Apical Osteocytes/TBV With Age. Mean osteocytes/TBV significantly decreased with age in apical bone ($r=-0.634$, $p=0.0027$).

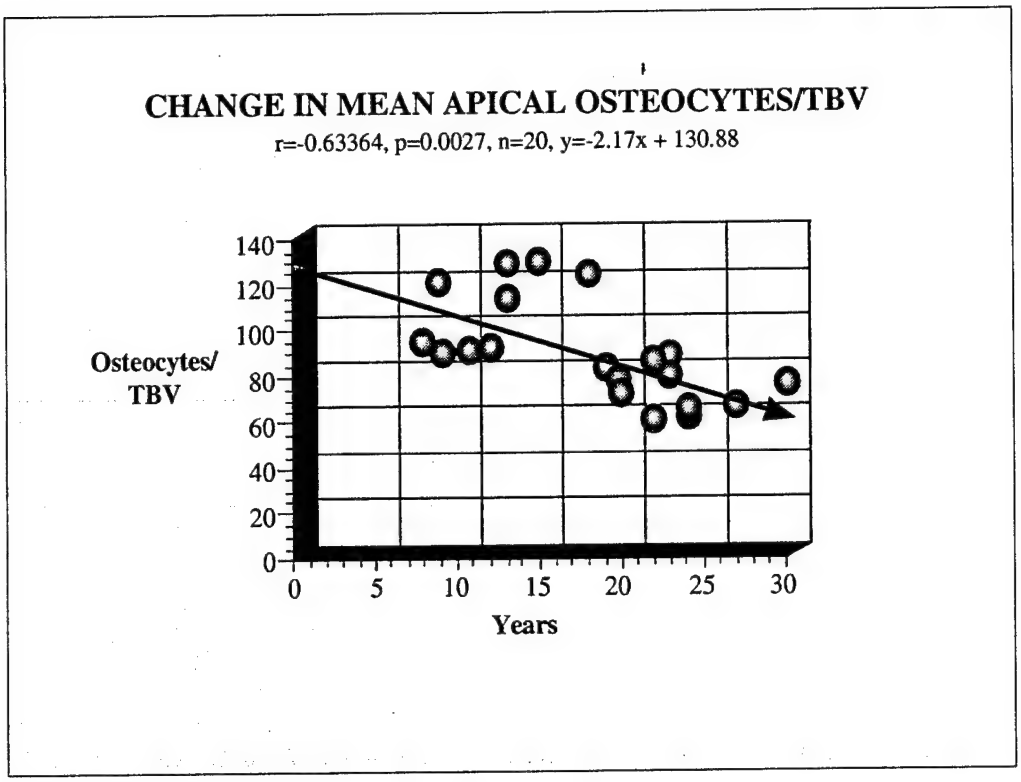

CHANGE IN MEAN APICAL OSTEOCYTES/TBV
r=-0.63364, p=0.0027, n=20, y=-2.17x + 130.88
Osteocytes/ TBV
140
120
100
80
60
40
20
0
0
5
10
15
20
25
30
Years

Figure 18. Mean Osteocytes By Age Group. Mean osteocytes were significantly different in old animals (18-30 years of age; HAE $\geq$ 54 years) compared to young animals (8-13 years of age; HAE $\leq$ 39 years) in apical bone but not in crestal bone.

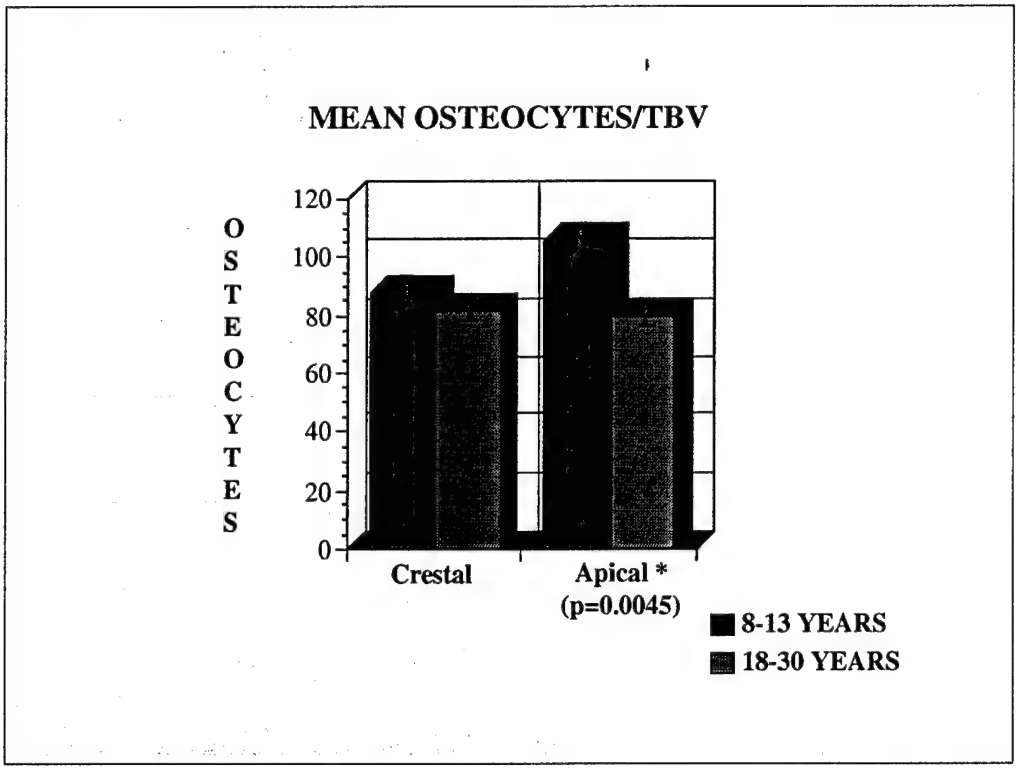
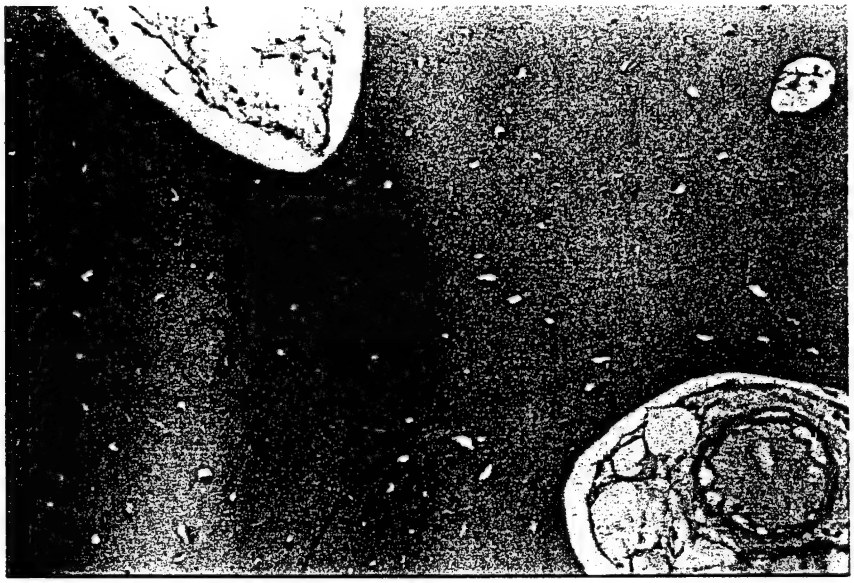
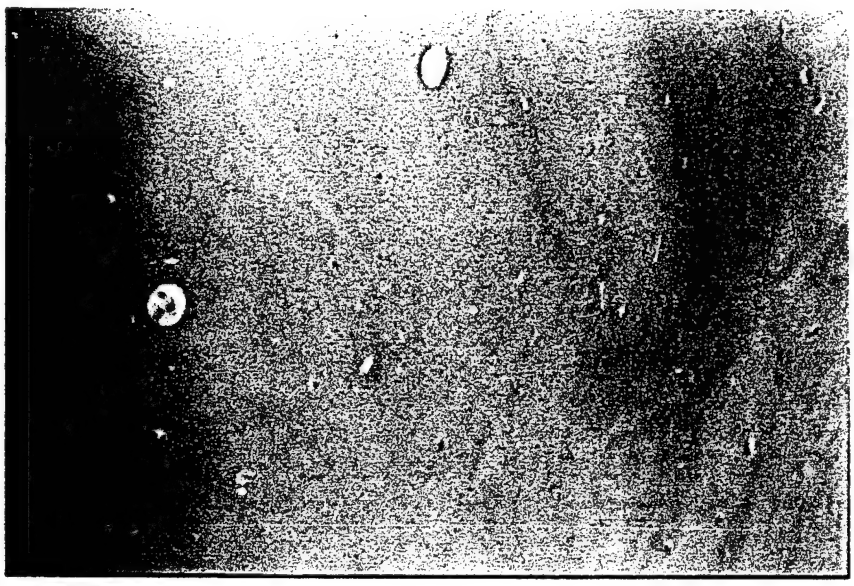

MEAN OSTEOCYTES/TBV
OSTEOCYTES
120
100
80
60
40
20
0
Crestal
Apical *
(p=0.0045)
8-13 YEARS
18-30 YEARS

Figure 19. Apical Bone in 8 Year-old Baboon. Highly cellular apical bone in a young animal (HAE 24 years). (HE x 215).

Figure 20. Apical Bone in 27 Year-old Baboon. Diminished apical osteocyte density in 27 year old animal (HAE 108 years). (HE x 215).





bone (Table 1); these parameters were not significantly different between age groups or categories of attachment loss (Tables 5 and 6).

5. Osteoblastic and Osteoclastic activity. QHA disclosed the finding that crestal osteoblastic surfaces decreased significantly with age ($r = -0.655$, $p = 0.0017$, Figure 21, Table 2). When animals were grouped by age, percent blastic surfaces significantly decreased in the older age group in crestal bone ($p = 0.047$, Figure 22, Table 5). Analysis disclosed that percent crestal blastic activity also decreased with increasing attachment loss ($r = -0.47$, $p = 0.035$, Table 3). Percent crestal blastic surfaces was also significantly correlated with interproximal distance (C4) ($r = 0.36$, $p = 0.028$, Table 4) at the site level. Osteoclastic activity was not significantly correlated with other morphometric parameters and not significantly different between age groups or categories of attachment loss.

B. <u>Analysis of Variance in Histologic Parameters.</u>

Certain histologic parameters proved to be significantly correlated not only with aging but also with other variables. For instance, crestal blastic surfaces significantly decreased with increasing animal age and increasing root proximity; however, it was also significantly associated with increasing attachment loss. A partial correlation was performed to determine which parameter provided the greatest source of variation (Table 7). Type III ANOVA demonstrated that the majority of the variability in crestal blastic activity is explained by aging ($r = -0.5$, $p = 0.0025$). Similarly, while attachment loss was significantly correlated with multiple parameters the variability in this parameter is primarily explained by age ($r = 0.48$, $p = 0.0035$) and root proximity ($r = -0.41$, $p = 0.014$).

Figure 21. Change in % Crestal Blastic Surfaces With Age. QHA revealed crestal osteoblastic surfaces decreased significantly with age ($r=-0.655$, $p=0.0017$)

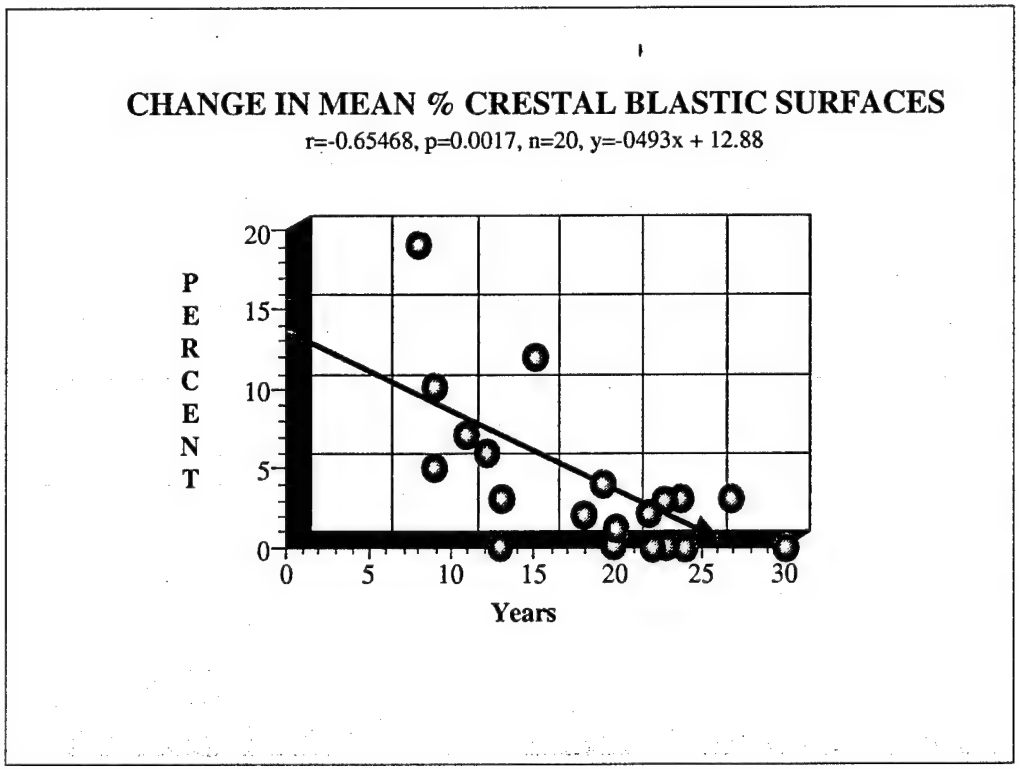

CHANGE IN MEAN % CRESTAL BLASTIC SURFACES
r=-0.65468, p=0.0017, n=20, y=-0493x + 12.88
PERCENT
20
15
10
5
0
0
5
10
15
20
25
30
Years

Figure 22. Percent Blastic Surfaces By Age Group. Percent blastic surfaces significantly decreased in old animals (18-30 years of age; HAE $\geq$ 54 years) compared to young animals (8-13 years of age; HAE $\leq$ 39 years) in crestal bone but not in apical bone.

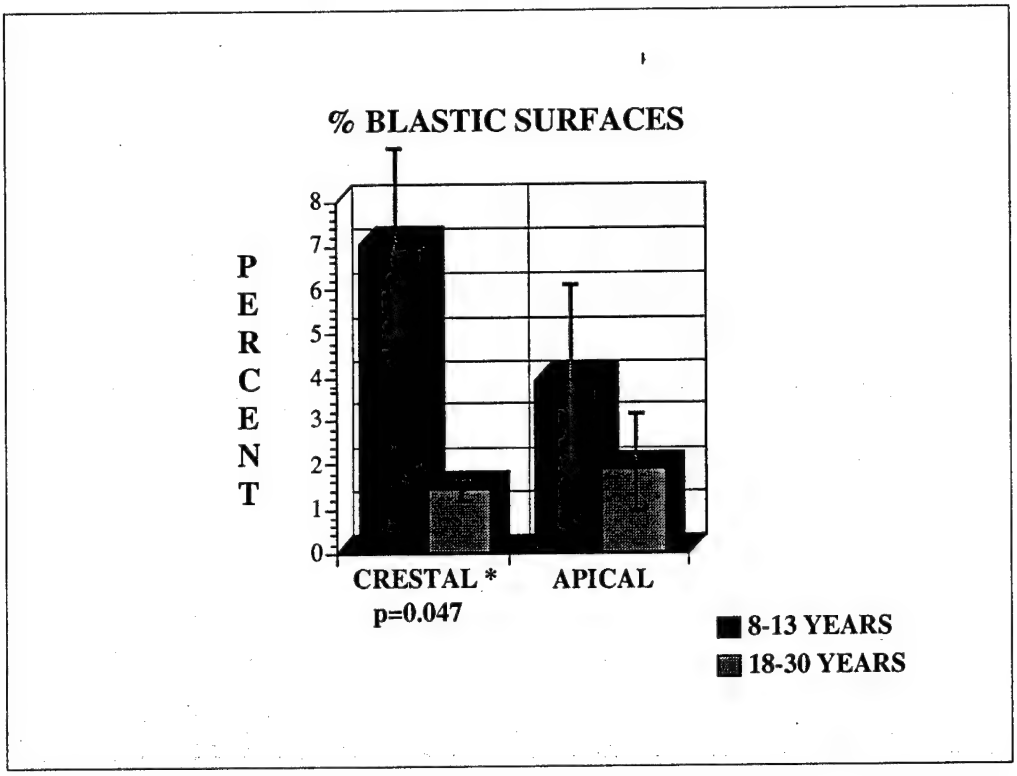

% BLASTIC SURFACES
PERCENT
0
1
2
3
4
5
6
7
8
CRESTAL *
p=0.047
APICAL
8-13 YEARS
18-30 YEARS

Table 7. TYPE III ANALYSIS OF VARIANCE: PARTIAL CORRELATION

Dependent Variable	Source of Variation	r	p
% Crestal Blastic Surfaces	Age	-0.5	0.0025 *
	Attachment Loss	0.02	0.8766
	Interproximal Dx (C4)	0.27	0.1104
Attachment Loss, mm	Age	0.48	0.0035 *
	% Crestal Blastic Surfaces	0.02	0.8766
	Interproximal Dx (C4)	-0.41	0.0147 *

* $p < 0.05$

IV. DISCUSSION AND SUMMARY

Attachment Loss In The Baboon Model

Animal models are utilized of necessity in scientific inquiry to test treatment effects or to study disease progression by means of invasive histological analysis not feasible in man (8, 16). As revealed in this cross-sectional sample of animals with HAE 24 to greater than 100 years of age, the baboon demonstrates limited spontaneous periodontal disease, even at advanced age; in all cases, mean histologic attachment loss was less than 3 mm. As such, the baboon may be better suited for studies involving standardized, experimentally-created defects (16). An obvious advantage afforded by the colony of some 3000 baboons housed at the Southwest Foundation for Biomedical Research in San Antonio is their known medical history (8) and pedigree. The reduced biologic variability afforded by this genetically-defined stock should qualify these animals as a good model system in the scientific context, as suggested by Selvig, rather than merely "convenient substitutes for human experimentation" (17).

Cross-sectional analyses provide baseline data about a population but are frequently plagued by inappropriate statistical assumptions or modeling resulting from selection of sites for sampling as well as the unit of analysis. In the present study, 21 baboons demonstrated an age-associated increase in histologic attachment loss confirming clinical probing data on 128 baboons from this population (11). The finding of age-associated attachment loss was significant using either the site or the subject as the unit of analysis. Tooth-associated correlation must be considered since measurements made from the same tooth or from adjacent teeth are more correlated than measurements from different teeth in the dentition (18). Ignoring within-subject or within-tooth correlation increases type 1 error, or falsely presuming a significant difference exists between study groups (rejecting a null

hypothesis). Gunsolley *et al.* (18) suggest measurement should be made at different teeth if limited measurements are taken from each subject. In the present study, histologic attachment measurements were made on 4 teeth, the right and left mandibular first and second molars. A standard site in the posterior mandible was necessarily sampled to avoid the wide site-to-site, anatomic variability which may confound bone morphometry (14). Interproximal molar sites were deliberately sampled to maximize the likelihood of locating disease since clinical probing data revealed high frequency of attachment loss in posterior sites (11).

Minimizing examiner error is likewise critical in cross-sectional investigation where the consistency of an examiner's singular measurement alters interpretation of population demographics. Cronbach's Coefficient Alpha (α) may be used to evaluate consistency of measurement; $\alpha > 0.7$ indicates good agreement between repeated measures. Intra-examiner reliability in this study was high evidenced by an $\alpha > 0.88$ for all parameters except one interproximal distance measure (C4) where the percentage error between repeated measures was < 1.6%. This error rate is minor given that a maximum error of 3% has been considered acceptable in previous morphometric investigation (19, 20).

The finding that increased attachment loss is related to aging in the baboon is consistent with numerous data from the human periodontium (21-23). Grossi *et al.* (24) reported that age was the single factor most associated with severity of increasing attachment loss; the relative risk (odds ratio) increased from 1.72 in humans 35-44 years of age to 9.01 at age 65-74. Brown *et al.* (25) recently reported that loss of periodontal attachment increases in prevalence, severity and extent with age. Explanations for this phenomenon have included the cumulative nature of periodontitis (26) or a shift in host response owing to physiological changes in aging (27).

While attachment loss is frequently associated with increasing age, loss of periodontal support need not be regarded as an obligatory consequence of aging (28). In study groups of Japanese (70-79 years of age) and Swedish individuals (60-65 years of age), Papapanou *et al.* reported 10% and 15% of sites per subject, respectively, revealed attachment loss $\leq$ 1 mm. It is interesting to note that just as in humans, attachment loss in the baboon is not necessarily a sequela of aging as 2 animals with HAE > 60 years displayed mean attachment loss < 0.5 mm.

Crestal resorption (ACL) in this study was significantly correlated with increasing attachment loss. While this was correlated with aging at the site level, crestal resorption was more so a function of periodontitis. The mean CT attachment dimension did not change with age or with increasing attachment loss. This dimension ranged from 0.7 - 2.9 mm and was the most consistent dentogingival junction value with a range narrower than values obtained for ACL or Attch Loss. The consistency of this measure, also the most constant dentogingival dimension in the human periodontium (29), further validates the appropriateness of the baboon model for histologic assessment of periodontal disease. Vacek has furthermore reported that the human CT attachment dimension does not strongly correlate with loss of attachment (13). Similarly in this baboon sample, the CT attachment dimension did not vary with advancing age or attachment loss.

Age-associated Changes In Alveolar Bone

This investigation represents an attempt to apply the same methodology used to evaluate systemic bone disease to the study of alveolar bone. Quantitative Histomorphometric Analysis provided evidence that significant age-related changes were evident in alveolar bone cellularity and activity including decreasing apical osteocyte density and crestal osteoblastic surfaces. These findings affirm early

observations by Tonna in mice that endosteal cell numbers decrease per surface and osteogenic precursors are reduced to an inactive morphology (30). Bernick *et al.* hypothesized that the observed calcification of nutrient canals in mandibles from aging humans may impair the transfer of calcium and phosphate to osteogenic cells and osteocytes (3). Reduced apical osteocytes in the current baboon alveolar bone sample may reflect a slower rate of bone formation in older animals whereby cells are less likely to be trapped in a mineralizing matrix. No differences in osteoclast number could be detected between young and old baboon groups. This finding contrasts with Hardt (31) who has reported a significant decrease in osteoclast number in interproximal bone of Wistar rats between 10 and 18 weeks of age.

Age-related changes in alveolar bone may have implications for regenerative therapy. The finding of reduced crestal blastic surfaces with age may suggest a decrease in component progenitor cells and thus diminished osteogenic potential in bone harvested from, or implanted into, elderly individuals. Evidence of reduced bone morphogenic protein activity in human bone matrix from older donors (after the 5th decade) may be related to these cellular changes (32). While not specifically designed to test the hypothesis, 2 clinical studies failed to disclose a significant difference in osseous regeneration based on the age of donor (33) or the graft recipient (34). The lack of a clinical effect from age in these 2 studies does not negate the possibility that donor bone from younger individuals may have enhanced regenerative capacity and thus be more desirable for grafting. In addition, regenerative therapy may be better-suited to younger patients. QHA revealed that osteoblastic activity also decreased with greater attachment loss; however, Type III ANOVA demonstrated that the majority of the variability is explained by aging. These findings suggest that the regenerative capacity of bone is impacted more so by age than by history of periodontal disease.

QHA fields were sampled at the mesiodistal midline of the interseptal bone in an effort to capture cancellous bone. Whether alveolar bone follows the same composition of cortical (85%) to cancellous bone (15%) as found in the skeleton remains to be determined. In human cadaveric material, Heins and Wieder (35) reported that cancellous bone exists when inter-root distance between first and second molars exceeded 0.5 mm. In the present study, minimum interproximal distance (C4) was greater than 0.58 mm at the site level (data not shown) except in one specimen where C4 was 0.27 mm and no bone existed. These data are consistent with the human periodontium where bone is not observed when inter-root distance is less than 0.3 mm (35).

Human alveolar bone may demonstrate decreasing density with advancing age similar to the decreasing bone mass observed in mandibular cortical bone with age (36, 37). Southard *et al.* (38) reported a significant radiographic difference in interproximal bone mass between younger women (mean age 24.4 years) and older women (mean age 74.4 years). Some authors suggest decreasing density may relate to systemic osteoporosis (5, 39-41) whereas other examiners have denied evidence of osteoporosis in biopsy material (4, 6, 37).

Age-related changes in alveolar bone density were not evident in this baboon sample. While other parts of the skeleton normally demonstrate demineralization with age, it is unclear whether alveolar bone is expressly protected from density changes evident with skeletal demineralization. Normally, absolute skeletal bone volume decreases some 40% from its peak at age 25 to age 70 (37). In the present study, alveolar bone was markedly resistant to loss of density. QHA revealed a trend for mean Total Bone Volume to decrease < 10% with age, but this change was not significantly different between the young versus the old group either for crestal or apical bone. Despite marked osteoporotic phenotype and significant histomorphometric loss of density in vertebrae of many elderly females from this

sample (12), no significant age-associated osteoporotic decrease in density was manifest in alveolar bone. Methodology or QHA parameters used were not sensitive enough to detect change in alveolar density, if existent. Different parameters may be necessary for analysis of alveolar bone than those used for systemic bone disease.

It is unclear whether anatomic factors such as the influence of root proximity may mask a systemic effect on alveolar bone density. Measurement of interproximal distance (C4), sampled at a standard distance (3.7 mm) from the most apical CEJ, disclosed the significant influence of root proximity on severity of attachment loss in these animals; attachment loss significantly increased as interproximal distance decreased. While other parameters were significantly correlated with attachment loss, the majority of variation was attributed to aging and the influence of root proximity. Although there was a slight trend for alveolar bone density to increase as interproximal distance decreased, root proximity was not significantly associated with TBV.

Skeletal osteoporosis or diminished bone mineral density has been associated with edentulism (43), increased tooth loss (44, 45) or periodontal disease (41, 46-48). Other investigators have found no significant difference in periodontal parameters based on osteoporosis or systemic bone mass (44, 49). Recently, however, von Wowern *et al.* (50) observed significantly increased mean attachment loss (3.65 mm) in osteoporotic women with reduced mandibular bone mineral content versus a normal cohort (2.86 mm). In the present study, alveolar bone density poorly correlated with increasing attachment loss. When animals were grouped according to severity of attachment loss there was no significant difference in mean TBV between the two categories of attachment loss. These data agree with Somerman (37) who suggested that an observed decrease in density of the alveolar crest can be associated with aging, independent of periodontal disease.

The interplay between periodontal disease and skeletal osteopenia was not tested in this study since skeletal bone density was not evaluated. Data from this non-human primate model suggest that alveolar bone appears to resist loss of density despite advancing age or periodontal disease. However, the lack of correlation between alveolar density (TBV) and attachment loss may reflect the limited nature of periodontal disease in this study sample. Whether alveolar bone is resistant to loss of density, perhaps due to the functional loading of teeth, is uncertain. Teeth in function may impart forces to bone which mask systemic density changes evident with aging. This notion is in line with the homeostatic effect of masticatory function which is observed to stimulate repair processes to resolve areas of ankylosis and regain normal periodontal width (51). Future investigation in this population will evaluate spinal QHA data to determine whether diminution of alveolar density is associated with systemic skeletal demineralization. Such investigation may uncover whether alveolar bone is subject to, or uniquely protected from, demineralization observed elsewhere in the systemic skeleton.

Conclusions:

Within the limitations of this study it appears:

1. The baboon histologically demonstrates naturally-occurring periodontitis, although not severe even at advanced animal age.

2. Quantitative age-associated changes reflecting diminished bone cellularity and activity are expressed in alveolar bone. These changes are more related to aging than to the cumulative effects of a localized inflammatory process.

Ness, K., Yuan, C., Waldrop, T., Fox, W., Aufdemorte, T. 1995. Age-related changes in alveolar bone of non-human primates. *Submitted to J Periodont Res.*

LITERATURE CITED

1. Roberts, W., Gonsalves, M. 1992. Aging of bone tissue. In: Disorders of Bone and Mineral Metabolism. Florida: Raven Press, Ltd., pp 83-93.

2. Van der Velden, U. 1984. Effect of age on the periodontium. *J Clin Periodontol* **11**:281-294.

3. Bernick, S., Sobin, S., Paule, W. 1983. Changes in the microvasculature and interstitium of aged human mandibles and maxillae. *Gerodontology* **2**:9-14.

4. Severson, J., Moffett, B., Kokich, V., Selipsky, H. 1978. A histologic study of age changes in the adult human periodontal joint (ligament). *J Periodontol* **49**:189-200.

5. Greenwell, H., Bissada, N. 1989. Factors influencing periodontal therapy for the geriatric patient. *Dent Clin North Am* **33**:91-100.

6. Grant, D., Bernick, S. 1972. The periodontium of ageing (sic) humans. *J Periodontol* **43**:660-667.

7. Meunier, P. 1988. Assessment of bone turnover by histomorphometry in osteoporosis. In: Osteoporosis: Etiology, Diagnosis and Management. New York: Raven Press, pp 317-332.

8. Schou, S., Holmstrup, P., Kornman, K. 1993. Non-human primates used in studies of periodontal disease pathogenesis: a review of the literature. *J Periodontol* **64**:497-508.

9. Page, R., Schroeder, H. 1982. In: Periodontitis in Man and Other Animals. New York: Karger, pp 192-204.

10. Avery, B., Simpson, D. 1973. The baboon as a model system for the study of periodontal disease: clinical and light microscopic observations. *J Periodontol* **44**:675-686.

11. Miller, D. 1994. Age, sex and genetics as determinants of periodontal disease in the baboon: a potential model for human disease. Masters Thesis. The University of Texas Health Science Center at San Antonio, San Antonio, Texas.

12. Aufdemorte, T., Fox, W., Miller, D., Buffum, K., Holt, R. 1993. A non-human primate model for the study of osteoporosis and oral bone loss. *Bone* **14**: 581-586.

13. Vacek, J., Gher, M., Assad, D., Richardson, A., Giambarresi L. 1994. Dimensions of the human dentogingival junction. *Int J Perio Rest Dent* **14**:154-165.

14. Revell, P. 1983. Histomorphometry of bone. *J Clin Pathol* **36**:1323-1331.

15. Fleiss, J., Park, M., Chilton, N. 1987. Within-mouth correlations and reliabilities for probing depth and attachment level. *J Periodontol* **58**:460-463.

16. Caton, J., Mota, L., Gandini, L., Laskaris B. 1994. Non-human primate models for testing the efficacy and safety of periodontal regeneration procedures. *J Periodontol* **65**:1143-1150.

17. Selvig, K. 1994. Discussion: animal models in reconstructive therapy. *J Periodontol* **65**:1169-1172.

18. Gunsolley, J., Williams, D., Schenkein, H. 1994. Variance component modeling of attachment level measurements. *J Clin Periodontol* **21**:289-295.

19. Kantor, M., Polson, A., Zander, H. 1974. Alveolar bone regeneration after removal of inflammatory and traumatic factors. *J Periodontol* **47**:687-695.

20. Caton, J., Nyman, S. 1981. Histometric evaluation of periodontal surgery III. The effect of bone resorption on the connective tissue attachment level. *J Periodontol* **52**:405-409.

21. Löe, H., Ånerud, Å., Boysen, H., Smith M. 1978. The natural history of periodontal disease in man. Tooth mortality rates before 40 years of age. *J Periodont Res* **13**:563-572.

22. Beck, J., Koch, G., Rozier, R., Tudor G. 1990. Prevalence and risk indicators for periodontal attachment loss in a population of older community-dwelling blacks and whites. *J Periodontol* **61**:521-528.

23. Douglass, C., Fox, C. 1993. Sectional studies in periodontal disease: current status and implications for dental practice. *Adv Dent Res* **7**:25-31.

24. Grossi, S., Zambon, J., Ho, A., Koch, G., Dunford, R., Machtei, E., Norderyd O., Genco, R. 1994. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *J Periodontol* **65**:260-267.

25. Brown, L., Oliver, R., Löe, H. 1990. Evaluating periodontal status of U.S. employed adults. *J Amer Dent Assoc* **121**: 226-232.

26. Waerhaug, J. 1966. Epidemiology of peridontal disease. In: World Workshop In Periodontitis. Ann Arbor, Michigan, p 194.

27. Bernier J. 1958. The role of organ systems and age in periodontal disease. *J Periodontol* **29**:247-253.

28. Papapanou, P., Lindhe, J. 1992. Preservation of probing attachment and alveolar bone levels in 2 random population samples. *J Clin Periodontol* **19**:583-588.

29. Gargiulo, A., Wentz, F., Orban, B. 1961. Dimension and relations of the dento-gingival junction in humans. *J Periodontol* **32**:261-267.

30. Tonna, E. 1976. Factors (aging) affecting bone and cementum. *J Periodontol* **47**:267-280.

31. Hardt, A. 1985. Maturational changes in alveolar bone of adult rats. *Gerodontology* 4:25-30.

32. Urist, M., Nillsson, O., Hudak, R., Huo, Y., Rasmussen J., Hirota, W., Lietze, A. 1985. Immunologic evidence of a bone morphogenic protein in the *milieu intérieur*. *Ann Biol Clin* **43**:755-766.

33. Sepe, W., Bowers, G., Lawrence, J., Friedlaender, G., Koch, R. 1978. Clinical evaluation of freeze-dried bone allografts in periodontal osseous defects - part II. *J Periodontol* **49**:9-14.

34. Machtei, E., Cho, M., Dunford, R., Norderyd, J., Zambon, J., Genco, R. 1994. Clinical, microbiological and histologic factors which influence the success of regenerative periodontal therapy. *J Periodontol* **65**:154-161.

35. Heins, J., Wieder, S. 1986. A histologic study of the width and nature of inter-radicular spaces in human adult pre-molar and molars. *J Dent Res* **65**:948-951.

36. von Wowern, N., Stoltze, K. 1978. Sex and age differences in bone morphology of mandibles. *Scand J Dent Res* **86**:478-485.

37. Somerman, M. 1984. Mineralized tissue in aging. *Gerodontology* **3**:93-99.

38. Southard, K., Southard, T. 1992. Comparison of digitized radiographic alveolar features between 20- and 70-year-old women. *Oral Surg Oral Med Oral Pathol* **74**:111-117.

39. Atkinson, J., Woodhead, C. 1968. Changes in human mandibular structure with age. *Archs Oral Biol* **13**:1453-1463.

40. Henrikson, P., Kjell, W. 1974. The mandible and osteoporosis (1). *J Oral Rehab* 1:67-74.

41. Manson, J. 1976. Bone morphology and bone loss in periodontal disease. *J Clin Periodontol* **3**:14-22.

42. Devlin, H., Ferguson, M. 1991. Alveolar ridge resorption and mandibular atrophy. A review of local and systemic factors. *British Dental Journal* **170**:101-103.

43. Daniell, H. 1983. Postmenopausal tooth loss. Contributions to edentulism by osteoporosis and cigarette smoking. *Arch Intern Med* **143**:1678-1682.

44. Kribbs, P. 1990. Comparison of mandibular bone in normal and osteoporotic women. *J Prosthet Dent* **63**:218-222.

45. Krall, E., Dawson-Hughes, B., Papas, A., Garcia, R. 1993. Skeletal bone density and tooth loss. *J Dent Res* **72** *(Special Issue)* Abstract No. 1700:316.

46. Groen, J., Menczel, J., Shapiro, S. 1968. Chronic destructive periodontal disease in patients with presenile osteoporosis. *J Periodontol* **39**:19-23.

47. Krook, L., Whalen, J., Lesser, G., Lutwak, L. 1972. Human periodontal disease and osteoporosis. *Cornell Vet* **62**:371-391.

48. Lutwak, L., Singer, F., Urist, M. 1974. Current concepts of bone metabolism. *Ann Intern Med* **80**: 630-644.

49. Elders, P., Habets, L., Netelenbos, J., van der Linden, L., van der Stelt, P. 1992. The relation between periodontitis and systemic bone mass in women between 46 and 55 years of age. *J Clin Periodontol* **19**:492-496.

50. von Wowern, N., Klausen, B., Kollerup, G. 1994. Osteoporosis: a risk factor in periodontal disease. *J Periodontol* **65**:1134-1138.

51. Wesselink, P., Beertsen, W. 1994. Repair processes in the periodontium following dento-alveolar ankylosis: the effect of masticatory function. *J Clin Periodontol* **21**:472-478.

VITA

Kay Lyn Messenger Ness was born in Glendale, California on 3 April 1957 to Cloyce and Rosemary Messenger. A Texas resident since 1965, she graduated magna cum laude from the University of Texas at Austin in 1979 with a Bachelor of Arts degree in Humanities. During her undergraduate years, she was elected to membership in the National English Honor Society and the Phi Beta Kappa national honor society. In 1983, Dr. Ness received her Doctor of Dental Surgery degree from the University of Texas Health Science Center at San Antonio, was elected to membership in the Omicron Kappa Upsilon honorary dental society and received the Delta Sigma Delta Scholastic Achievement Award. Dr. Ness was commissioned in 1983 as an officer in the United States Air Force. In 1984, following completion of a General Practice Residency at Davis-Monthan Air Force Base in Tucson, Arizona, Dr. Ness was assigned as a general dental officer at the United States Air Force Academy, Colorado Springs, Colorado. After a 2-year tour at Clark Air Base, Republic of the Philippines, she was reassigned in 1991, to Lackland Air Force Base, Texas. Dr. Ness entered graduate training in Periodontics in June, 1992 at Wilford Hall Medical Center and the University of Texas Health Science Center at San Antonio. Her previous journal publications include *Effect of function and rest on the amplitude of the TMJ click*; J Oral Rehab 14:261-266, 1987. Dr. Ness married Air Force orthodontist, Colonel Charles F. Ness on 16 October 1986.